

## Left anterior descending coronary artery obstruction *Clinical, electrocardiographic, and angiographic correlates*

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Seventy-six patients with severe (greater than 80%) occlusive left anterior descending coronary artery disease by coronary angiography were examined for the electrocardiographic characteristics of this disease in the presence (group A 59 patients) or the absence (group B 17 patients) of anterior wall asynergy (akinesis or dyskinesis). The incidence of clinically documented anterior myocardial infarction in these two groups of patients was examined. The collateral circulation to the left anterior descending coronary artery was also examined in the groups of patients with and without anterior wall asynergy. Thirty-eight of 59 (64%) patients with anterior wall asynergy (group A) showed electrocardiographic signs of anterior myocardial infarction, 17 per cent showed probable electrocardiographic signs of anterior myocardial infarction and 19 per cent showed no electrocardiographic signs. None of the 17 patients without anterior wall asynergy (group B) showed electrocardiographic signs of anterior myocardial infarction. In group A 74.6 per cent had documented clinical evidence of previous anterior myocardial infarction, whereas in group B none had clinical evidence of previous anterior myocardial infarction. Collateral filling of the distal left anterior descending coronary artery was seen in 71 per cent of group A and 100 per cent of group B patients. There was a significantly higher incidence ( $P=0.02$ ) of collateral filling in the patients without electrocardiographic evidence of definite anterior myocardial infarction (93% of 28 patients), than in those who showed definite electrocardiographic evidence of anterior myocardial infarction (66% of 38 patients).

It is concluded that severe occlusive left anterior descending coronary artery disease with anterior wall myocardial asynergy is usually associated with electrocardiographic signs of anterior myocardial infarction, whereas equally severe left anterior descending coronary artery disease without anterior wall asynergy is rarely associated with electrocardiographic abnormalities of anterior myocardial infarction. Severe left anterior descending coronary artery obstruction without electrocardiographic and angiographic evidence of anterior myocardial infarction is usually associated with collateral circulation to the left anterior descending coronary artery and collateral circulation to the left anterior descending coronary artery is present less frequently when obstruction is associated with anterior myocardial infarction.

The theoretical and empirical basis for the association between abnormal Q waves and myocardial scars has been well established (Wilson *et al.*, 1933; Pardee, 1930; Fenichel and Kugell, 1931; Myers, Klein, and Hiratzka, 1949; Goldberger, 1945). Several investigators have looked at the classical QRS electrocardiographic abnormalities and attempted to correlate these with the presence, site, or extent of myocardial scar formation or coronary artery

disease at necropsy (Horan, Flowers, and Johnson, 1971; Myers, Klein, and Stofer, 1948) or at angiography (Hilsenrath *et al.*, 1972; Friesinger and Smith, 1972; Hamby, Gupta, and Young, 1973; Williams *et al.*, 1973; Bodenheimer, Banks, and Helfant, 1975; Miller *et al.*, 1974), with varying results. In individual patients the resting electrocardiogram appears to be an inaccurate predictor of the extent of coronary artery disease unless classical transmural myocardial infarction changes

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are present (Benchimol *et al.*, 1973). Pathological Q waves on the electrocardiogram correlate very closely with localized asynergy on left ventricular cineangiography (Williams *et al.*, 1973; Miller *et al.*, 1974). This appears to be true, especially with anterior myocardial infarction as opposed to inferior wall myocardial infarction (Williams *et al.*, 1973; Miller *et al.*, 1974).

We have previously reported on the correlation of inferior wall myocardial infarction with the presence and extent of pathological Q waves in leads II, III, and aVF in patients with high grade obstructions of the right coronary artery (Shettigar *et al.*, 1974). It is the purpose of this study to: (1) evaluate electrocardiographic abnormalities associated with complete or severe obstruction of the left anterior descending coronary artery in patients with or without associated anterior wall asynergy; (2) study the incidence of clinically documented anterior myocardial infarction in these two groups of patients; and (3) examine the possible role of coronary collaterals to the left anterior descending coronary artery.

### Methods

The coronary arteriograms and left ventricular cineangiograms of all patients who underwent these studies during the last three years at Palo Alto V.A. Hospital were reviewed. All patients who had significant disease (greater than 80% obstruction) of the left anterior descending coronary artery were selected for this study. Excluded were those patients who had had previous cardiac surgery or a left bundle-branch block pattern on the electrocardiogram, and those on whom inadequate clinical data were available, or cineangiograms were inadequate for interpretation because of atrial fibrillation or ventricular premature beats. Patients with congestive heart failure were also excluded. All patients included had disabling but stable angina pectoris and were considered to be candidates for saphenous vein aortocoronary bypass surgery. Coronary arteriography was usually done by the Judkins technique (Judkins, 1967) but in a few cases the Sones technique was used (Sones and Shirley, 1962). Multiple views of each coronary artery were obtained. Two subtotal (>80%) occlusions in close proximity or a subtotal and a total occlusion in close proximity or a long segment of narrowing have been considered as a single lesion. Nitroglycerin was not routinely used to enhance collateral vessel visualization. Collateral visualization of the left anterior descending coronary artery was graded as either present (left anterior descending coronary artery distal to the obstruction well visualized and at least of

the calibre required for saphenous vein aortocoronary bypass surgery) or absent (no or only poor collateral filling of the left anterior descending coronary artery distal to the obstruction). All left ventricular cineangiograms were done during held inspiration in the right anterior oblique position, and usually 10 to 15 minutes after the coronary arteriograms. A careful inspection was made of any localized contraction abnormalities (Herman *et al.*, 1967) over the anterior or apical areas of the left ventricle by two independent observers with good agreement in all cases. Only definite localized akinetic or dyskinetic left ventricular wall motion was considered as asynergy in this study. If the contraction abnormality was localized to the apex without involvement of the anterior wall and there was an associated contraction abnormality over the diaphragmatic area, the patient was excluded from the study since conceivably an obstruction of the right coronary artery with a long posterior descending branch could give a contraction abnormality of the apical and diaphragmatic surface. None of the patients who showed left ventricular anterior wall asynergy experienced angina pectoris during left ventricular angiography. In one patient a satisfactory left ventricular cineangiogram was not obtained; in this patient scarring over the left ventricular anterior wall was found at the time of surgery.

Seventy-six patients with total or severe occlusive disease of the left anterior descending coronary artery met our criteria and were included in the study. They were subdivided into two groups: Group A showed definite localized anterior wall asynergy on left ventricular angiography. Group B showed no anterior wall asynergy.

The electrocardiograms, recorded within 5 days of the angiograms, were then examined for evidence of healed anterior wall myocardial infarction using the following criteria:

- (1) Definite anterior wall myocardial infarction: Q waves (greater than 0.04 s duration) or QS deflections in (a) V1 to V2 or V4—anteroseptal wall myocardial infarction; (b) V2 or V3 to V4—strictly anterior wall myocardial infarction; (c) V3 or V4 to V6—anterolateral wall myocardial infarction.
- (2) Possible anterior wall myocardial infarction: (a) poor R wave progression across the praecordium (V1 to V6); (b) progressive decrease in the R wave across the praecordium (V1 to V5); (c) small Q waves, less than 0.04 s duration (V2 to V4 or V5).

The incidence of previous anterior myocardial infarction was examined in the two groups of patients. The diagnosis of previous anterior

TABLE 1 *Angiographic findings in 76 patients with left anterior descending coronary artery obstruction*

Groups	No. with left ant. desc. coron. art. disease	No. with 2-vessel disease	No. with 3-vessel disease	Previous anterior myocardial infarction documented in hospital
A	59	27	18	44
B	17	5	9	0
Total No.	76	32	27	44

Group A=Anterior wall asynergy present.

Group B=Anterior wall asynergy absent.

TABLE 2 *Distribution of coronary arterial lesions in 76 patients with left anterior descending coronary artery obstruction*

<i>Lesions in left anterior descending coronary artery</i>				
<i>Degree of narrowing</i>	<i>Group</i>	<i>Proximal</i>	<i>Middle</i>	<i>Distal</i>
100%	A	39	5	2
	B	13	2	1
Between 80% and 100%	A	11	2	—
	B	1	—	—
<i>Lesions in other coronary arteries of greater than 80% obstruction:</i>				
<i>Group</i>	<i>Right coronary</i>	<i>Left circumflex</i>	<i>Left main</i>	
A	46	33	2	
B	12	9	2	

myocardial infarction was made on the basis of a clear history of myocardial infarction accompanied by typical acute electrocardiographic changes and a rise in serum enzymes.

### Results

Table 1 shows the clinical profile of our 76 patients. They were all men, ranging in age from 26 to 66 years, with a mean of 49 years. Table 2 shows the results of coronary arteriography. As can be seen, 62 patients (81.6%) had 100 per cent obstruction of the left anterior descending coronary artery, usually in the proximal third, while 14 patients (18.4%) had a subtotal obstruction (greater than 80%). Associated disease of the right coronary artery was noted in 58 patients, of the left circumflex in 42 patients, and of the left main coronary artery in 4 patients. Of 59 patients in group A, 44 (74.6%) had history, electrocardiographic, and laboratory evidence of anterior wall myocardial infarction.

The electrocardiographic changes are summarized in Table 3. In group A 38 patients (64%) show changes of a definite and 10 patients (17%) of a possible myocardial infarction over the anterior wall. In 19 per cent of group A patients and 100 per cent of group B patients there were no electrocardiographic changes of a previous myocardial infarction.

Good collateral filling of the distal left anterior descending was noted in 100 per cent of group B

patients (Table 4). In group A 66 per cent of those with a definite myocardial infarction, 80 per cent of those with a possible myocardial infarction, and 82 per cent of those with no myocardial infarction on electrocardiogram had collateral filling of the distal left anterior descending coronary artery. Fig. 1 and 2 illustrate the different angiographic appearances in groups A and B and correlate the coronary arteriographic findings with left ventriculography. If one looks at all of the patients with 100 per cent obstruction of the left anterior descending coronary artery (78% of group A and 94% of group B patients) it can be seen that there was collateral filling of the left anterior descending coronary artery in 80.4 per cent of group A and 100 per cent of group B patients (Table 5).

### Discussion

Previous studies have attempted to correlate the various electrocardiographic changes with post-mortem findings (Fenichel and Kugell, 1931; Myers *et al.*, 1949; Horan *et al.*, 1971). More recently, others have tried to correlate electrocardiographic changes with the coronary arteriographic and left ventricular angiographic changes (Hilsenrath *et al.*, 1972; Friesinger and Smith, 1972; McConahay *et al.*, 1970; Williams *et al.*, 1973; Bodenheimer *et al.*, 1975; Miller *et al.*, 1974). Usually the approach has been to start with the electrocardiogram and then to examine the post-

mortem or arteriographic findings. The approach in this study is somewhat different and is similar to that of Hamby *et al.* (1973) in selecting patients for the study on arteriographic criteria and then looking for clinical and electrocardiographic correlates. Several investigations have shown good correlation between scar formation over the left ventricular anterior wall at necropsy or anterior or apical left ventricular asynergy on left ventricular cineangiography and either pathological Q waves over various combinations of leads V1 to V6 (Horan *et al.*, 1971; Williams *et al.*, 1973; Bodenheimer *et al.*, 1975; Miller *et al.*, 1974) or significant left anterior descending coronary artery disease (Williams *et al.*, 1973).

Our patients generally had severe coronary artery disease with an average of 2.4 severe lesions (80% or greater) per patient (Table 2). Many patients had associated inferior wall infarction. The effect of these associated coronary lesions and inferior wall myocardial infarctions on the electrocardiographic

changes of anterior wall myocardial infarction is not known but must be considered. Of note, however, is the fact that the electrocardiographic pattern in V1 to V6 in 10 patients with isolated left anterior descending coronary artery disease and no inferior infarction did not differ significantly from those with associated coronary artery disease or inferior infarction.

The resting electrocardiogram has been found to be a poor predictor of the extent of coronary artery disease (Benchimol *et al.*, 1973). Most workers have found that in patients with electrocardiographic abnormalities indicating definite myocardial infarction there is good correlation between the site of coronary artery disease predicted from the electrocardiogram and the area of left ventricular asynergy (Williams *et al.*, 1973). In this study, looking at equally severe disease of the left anterior descending coronary artery, patients who showed anterior wall asynergy (group A) were much more likely to have electrocardiographic abnormalities of

TABLE 3 *Electrocardiographic findings in patients with left anterior descending coronary artery obstruction*

Groups	Total	Anterior myocardial infarction					
		None	Possible	Definite	Antero-septal	Strictly anterior	Antero-lateral
A	59	11 (19%)*	10 (17%)	22 (37%)	6 (10%)	10 (17%)	
B	17	17 (100%)					

\*7/11 patients had 100 per cent occlusion of the left anterior descending coronary artery.

TABLE 4 *Collateral filling of distal left anterior descending coronary artery*

Group A	Total no.	No. with collateral filling	Per cent	No. without collateral filling	Per cent
Electrocardiogram					
Definite MI	38	25	66	13	34
Possible MI	10	8	80	2	20
No MI	11	9	82	2	18
Total	59	42	71	17	29
Group B					
No MI	17	17	100	0	0

MI—myocardial infarction.

TABLE 5 *Collateral filling of distal left anterior descending coronary artery in patients with total left anterior descending coronary artery obstructions*

	Group A	Collateral filling	Group B	Collateral filling
100% occlusion				
Proximal third	39	36	13	13
Middle third	5	1	2	2
Distal third	2	0	1	1
Total	46	37 (80.4%)	16	16 (100%)

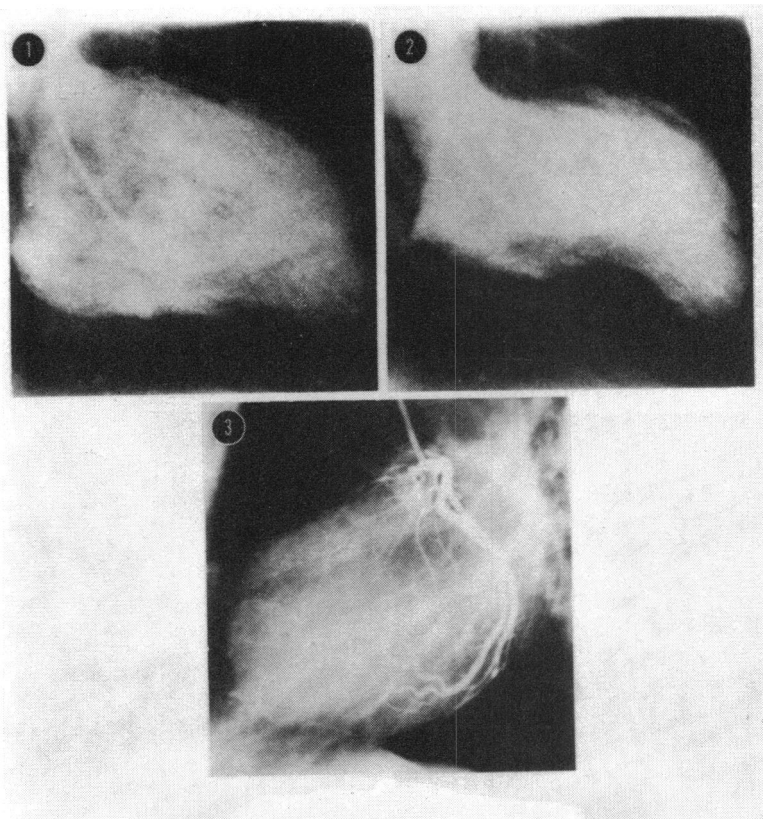


FIG. 1 Typical example of patient from group A, showing enlarged left ventricle (1) in diastole and (2) in systole. Note extensive and severe asynergy of apical and anterior wall segments. Left coronary artery injection (3) in lateral projection, showing proximal total occlusion of left anterior descending coronary artery. No collaterals seen from either left or right coronary artery injections.

a definite (64%) or possible (17%) myocardial infarction than patients without associated anterior wall asynergy (0%) (group B). A total of 11 patients (19%) with left ventricular anterior wall asynergy had no electrocardiographic evidence of anterior myocardial infarction. These results are in agreement with those of Miller *et al.* (1974) who found that 24 per cent of patients with left ventricular contraction abnormalities had no evidence of myocardial infarction on their electrocardiograms and with those of McConahay *et al.* (1970) who found that myocardial infarction diagnosed by electrocardiogram or vectorcardiogram is not a consistent finding in patients with ventricular contraction abnormalities. Miller *et al.* (1974), however, also included patients with hypokinesis whereas our group A patients included only those with akinesis or dyskinesis of the left ventricle. Our study also

differs from previous published reports (Williams *et al.*, 1973; Bodenheimer *et al.*, 1975; Miller *et al.*, 1974) in that we have investigated the correlation between collateral circulation to the obstructed left anterior descending coronary artery and the left ventricular contraction abnormalities and electrocardiographic signs of anterior myocardial infarction. Furthermore, the incidence of clinically documented anterior myocardial infarction was investigated in both group A and B patients. Our patient population, patient selection, and methods are also slightly different.

Collateral circulation to the left anterior descending coronary artery is dependent on the absence of severe occlusive disease or thrombosis in the distal portion of the vessel. Extensive anterior myocardial infarction may be followed by thrombosis of the left anterior descending coronary artery distal to the

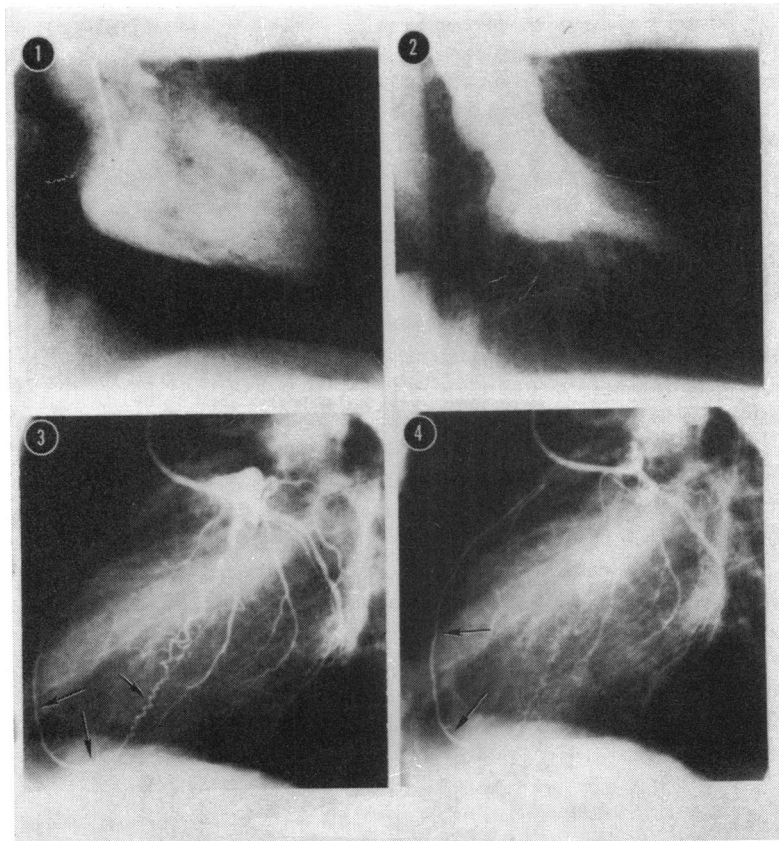


FIG. 2 Typical group B patient showing normal left ventricular contraction (1) in diastole and (2) in systole. Coronary arteriography in lateral projection showed total proximal occlusion of left anterior descending coronary artery but with early retrograde filling via collaterals around apex (3) indicated by arrows. Later frame from same injection (4) shows excellent retrograde filling back to site of proximal stenosis.

obstruction, and in such patients collateral filling of distal segment of the vessel does not occur; whether this is an important factor in group A patients is not known. In this study, collateral filling of the left anterior descending was noted in 71 per cent of group A patients and 100 per cent of group B patients. In the total group a significantly higher incidence ( $P=0.02$ ) of collateral filling was observed in the patients who had no definite electrocardiographic signs of anterior myocardial infarction (93% of 28 patients) compared with the patients who showed electrocardiographic signs of definite anterior myocardial infarction (66% of 38 patients) (Table 4). There is disagreement on the significance of arteriographically shown coronary collaterals. Some feel that their presence is merely an expression of the severity of coronary artery disease (Hamby

*et al.*, 1973; McConahay *et al.*, 1970; Miller *et al.*, 1972) and no protection against myocardial ischaemia as measured by electrocardiographic stress testing (Miller *et al.*, 1972; Helfant, Vokonas, and Gorlin, 1971). Others noted that the presence of collateral vessels in severe coronary artery disease is more likely to be associated with a normal electrocardiogram (Gensini and DaCosta, 1969), can enhance myocardial contractility in dogs (Cohen *et al.*, 1973), and can improve function in asynergic segments in the absence of Q waves in man after nitroglycerin (Banka, Bodenheimer, and Helfant, 1974), and may decrease mortality in man (Helfant *et al.*, 1971). In this study no significant difference was noted in the severity of coronary artery disease (number of arteries obstructed per patient) between group A and B patients, or between patients

with and without definite electrocardiographic signs of anterior myocardial infarction. However, from the data one may conclude that the development of collateral circulation in association with left anterior descending coronary artery obstruction probably prevented the occurrence of acute anterior myocardial infarction. This conclusion is supported by animal studies (Blumgart *et al.*, 1950; Blum, Schauer, and Calef, 1938).

The effect of coronary artery dominance on the presence of anterior wall contraction abnormalities as well as on the electrocardiographic signs of anterior myocardial infarction was considered, but no significant correlation was noted. Failure to infarct and thus, failure to show anterior wall asynergy in 17 patients (group B) may in certain cases be attributed to a large posterior descending coronary artery or branches of left coronary artery supplying the territory of the left anterior descending coronary artery, especially when the latter is small. This, however, was not so in any patient in this study where most patients had severe 2- or 3-vessel disease.

In summary, in examining the significance of high grade left anterior descending coronary artery obstructions, it has been shown that left ventricular contraction abnormalities are much more likely to be associated with electrocardiographic criteria of a definite myocardial infarction. Collateral filling of the distal left anterior descending coronary artery is less frequently seen when definite myocardial infarction changes are noted on electrocardiogram and localized anterior and/or apical contraction abnormalities are seen on the left ventricular cineangiogram.

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